Intra-abdominal Ultrasound

Intra-abdominal Hypertension and Abdominal Compartment Syndrome

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KEYWORDS

• Ultrasound • Abdominal compartment syndrome • Intra-abdominal hypertension
• Point of care ultrasound • Intra-abdominal pressure • VExUS

KEY POINTS

- Abdominal compartment syndrome (ACS) can be fatal and rapid assessment and management is imperative.
- The ability to use ultrasound in the diagnosis and management of ACS is evolving.
- Ultrasound can be used to calculate renal artery indices and inferior vena cava diameters, which correlate with intra-abdominal pressures.
- Novel concepts of ultrasound in ACS are rapidly changing the paradigm of identification and treatment of ACS.

INTRODUCTION

The abdomen and pelvis together comprise a compartment with distinct anatomic boundaries. Its fixed location and closed anatomy put all its contents at the physiologic dependence of its pressure. The normal intra-abdominal pressure (IAP) is between 2 and 7 mm Hg. In this environment, the organs, vessels, and components maintained in the cavity are at physiologic pressure.[1]

Abnormalities of the IAP can result in significant pathology, ultimately resulting in compartment syndrome. Compartment syndrome was first described in the 1800s in the extremities. When the gap between tissue pressure and perfusion/flow into the space decreases, tissue viability is threatened. In the early 1900s, Emerson began to describe this relationship as it pertains to the abdominal cavity. While important work garnered interested and attention to the concept of IAP and the risk for pathology, several decades passed before this topic resurfaced as it relates to the treatment of abdominal wounds during war. It was not until the 1980s that a formal description of

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Abbreviations	
IAH	intra-abdominal hypertension
IAP	intra-abdominal pressure
IVC	inferior vena cava
POCUS	point of care ultrasound
RI	resistive index
VExUS	venous excess ultrasound score
WSACS	World Society of the Abdominal Compartment Syndrome

abdominal compartment syndrome (ACS) was proposed by Kron, Harman, and Nolan.[2] Stated succinctly, "IAPs below 20 mm Hg in a postoperative patient in the absence of rapid blood loss or renal insufficiency are an indication for continued observation. An IAP above 25 mm Hg in a postoperative patient with an adequate blood volume and a low urinary output is an indication for abdominal re-exploration and decompression."[3] Simply put, abdominal compartment syndrome is defined as intra-abdominal hypertension (IAH) resulting in end organ failure. To further define this disease process as well as promote research and education, the World Society of the Abdominal Compartment Syndrome (WSACS), now termed the Abdominal Compartment Society, was formed in 2004. The society initially created and has subsequently updated definitions, publishing the first formal definitions in 2006 defining pathologic changes in the IAP.

The WSACS has worked diligently to create a standardized taxonomy for defining IAH and ACS. Most recently, IAH is defined as IAP $\geq$12 mm Hg. Beyond that, various grades (I-IV) of IAP are defined (**Table 1**). ACS is defined as IAH greater than 20 mm Hg, which is associated with new organ dysfunction/failure.[4–6] Elevations in IAP and eventual ACS have far reaching consequences for the patient and affect essentially every organ system, including cardiac, respiratory, renal, and even neurologic function. Further, ACS can be classified as primary or secondary. Primary ACS occurs when there is a direct source of IAP within the abdominal cavity (trauma, infection, pancreatitis, etc.). Secondary ACS results as a result of extra-abdominal factors such as massive resuscitation, heart failure, etc. This article will focus on the recognition, diagnosis, and management of compartment syndrome, with a unique focus on the use of ultrasound to diagnose the condition.

PATHOPHYSIOLOGY

Overview

Elevation of IAP compromises nearly all vital organ systems. While critically ill patients have been found to have mild elevations in IAP of 5 to 7 mm Hg, patients with higher elevations are at risk for complications. It was originally thought that when the abdominal perfusion pressure (mean arterial pressure minus IAP) becomes inadequate then ACS ensues. However, Olofsson demonstrated that this may not be a direct, linear

Table 1
Intra-abdominal hypertension grading scheme

Grade	Intra-abdominal Pressure(mm Hg)
I	12–15
II	16–20
III	21–25
IV	>25

affect equally applied to all organ systems. Using a swine model, they found that small bowel mucosal blood flow was better preserved in comparison to other organ systems, suggesting a form of autoregulation.[7] While much of the focus for IAH and ACS is on the pressure, there is certainly a cellular component that prompted Malbrain and colleagues to coin the terms acute bowel injury and acute intestinal distress syndrome. Acute bowel injury results in capillary leak and formation of edema. This process of cellular and cytokine activation may lead to IAH, which results in additional bowel insults, leading to acute intestinal distress syndrome. The authors correlate this to the concept of acute respiratory distress syndrome (ARDS), but this physiologic blueprint that is widely accepted for ARDS has been slow to be applied to the gastrointestinal tract.[8]

Importantly, the effects of ACS reach far beyond the bowel and even the abdomen. Cardiovascular collapse may occur due to impairment in venous return to the right ventricle. Additionally, respiratory failure can result from upward compression on the diaphragm and subsequent increased airway pressures and decreased vital capacity. Renal function decreases from impaired perfusion. Increasing intracranial pressures secondary to increased IAP affects neurologic function. These alterations in physiology from ACS provide targets for ultrasound utilization in the diagnosis of ACS.

Cardiovascular

Preload reduction occurs via upward displacement of the diaphragm and the direct impact of pressure on the inferior vena cava, which decreases venous return. Afterload is similarly increased through direct pressure increasing systemic vascular resistance throughout. Contractility is affected resulting in impaired function of both the right and left heart. Together, these effects decrease cardiac output. The right ventricle is very susceptible to these changes and the result is a decreased right ventricular ejection fraction.[9,10] There is reduction of left ventricular compliance and contractility, as demonstrated in an animal model, which confirmed impaired left ventricular relaxation with IAH. Collectively, these changes result in a flattening and right/downward shift of the Frank-Starling curve. From a treatment standpoint, this means that a small increase in preload will have a very small change in stroke volume.[9] Without addressing ACS, additional large volume fluid resuscitation may be deleterious, a concept known as “futile crystalloid preloading cycle.”[11] This concept makes ultrasound an attractive tool to assess volume statues and response to fluids in the setting of IAH.

Respiratory

Respiratory compromise, similar to cardiovascular effects, is multifactorial. Due to the close proximity of the thorax to the abdominal compartment, there is a direct transmission of pressure to the intrathoracic cavity in ACS.[12] The reported amount of pressure transmitted varies, but this can ultimately result in ARDS. There is an increase in alveolar pressure and dead space and a decrease in functional residual capacity and compliance, as well as an increase in lung edema, theorized secondary to pressure induced neutrophil activation. These alterations in physiology result in a requirement for ventilator changes to counteract these changes in physiology. Overcoming this usually requires an increase in positive end expiratory pressure, which can further worsen lung injury.[9]

Diaphragm changes also occur as a result of increased IAP. Normally, the thickness of the diaphragm increases as the muscle is shortened upon inspiration, but if there is increased pressure from the abdomen then the diaphragm cannot shorten as much.

This change of the diaphragm muscle has been studied as a target for ultrasound diagnosis of ACS and will be discussed below. The other respiratory changes described herein do not usually result in an ultrasound target for diagnosis or management, but it would have been incomplete to not discuss all of these physiologic changes.

Renal

The effects on the renal system are similarly complex and not fully understood as there are chemical components in addition to the physical ones. Decreased urine output has been reported in IAH as early as the 1800s and is not a new phenomenon.[13] Studies have demonstrated decreased renal blood flow as a direct result of elevated IAP. There is an increase in catecholamines, renin, angiotensin, and inflammatory cytokines that all likely contribute to renal dysfunction. And lastly, as previously mentioned, there is a reduction in cardiac output that also plays a significant role in renal failure.[14] While much of the focus on renal function is on arterial inflow, elevated venous outflow pressures can also have significant effects on renal function. These effects on the kidney play a very important role in the ability to evaluate flow and resistive indices using ultrasound in the diagnosis of ACS and will be discussed in more depth.[15]

Gastrointestinal

Elevations in IAP have been shown to have direct effects on intestinal blood flow. In a swine model, direct measurements of mesenteric arterial blood flow demonstrated a significant decrease with increasing IAP. There was a direct correlation of increasing ischemia with increasing pressure.[16] However, another animal study did not find evidence of intestinal ischemia with increased IAP.[17] It is logical that increased pressure within the abdomen directly translates to the bowel and intra-abdominal contents, increasing the risk of ischemia. However, given mixed studies, this should be considered cautiously, bowel perfusion autoregulation is certainly in play.

IAH can also lead to decreased venous return from compression of the mesenteric vessels resulting in ileus, intestinal edema, and ascites,[18] These changes from increases in IAP result in a vicious cycle of furthering bowel edema and IAP[19] These changes in bowel activity, bowel wall edema, and ascites are all targets that can be assessed on point of care ultrasound.[20]

Central Nervous System

Cerebral perfusion is directly related to IAH, as impaired cerebral venous outflow results in elevated intracranial pressures. Prolonged IAH can lead to significant reductions in cerebral perfusion pressure due to elevated ICP and can have effects on the mental status of critically ill patients.[19,21] Ultrasound has been shown to be effective in intracranial pressure estimation, but is outside the scope of this article.[22]

Diagnosis

Early recognition and diagnosis of ACS is of paramount importance to mitigate its deleterious effects. Subtle changes in urine output, increased ventilator/airway pressures, abdominal distention, and hypotension are all signs of IAH and ACS. Interestingly, clinical examination alone is not reliable with the diagnosis.[23] The traditional, gold standard for diagnosis of IAH and ACS is direct measurement of the IAP. While various techniques have been described, the most accepted method is the measurement of bladder pressure, as originally described by Kron.[3] This technique is well described and is beyond the scope of this article.

The WSACS only recommends the use of abdominal ultrasound in their management algorithm to identify space occupying lesions.[5] However, more recently, point of care ultrasound (POCUS) has become an increasingly advanced diagnostic tool and is reported to be feasible in the diagnosis and management of IAH and ACS and will be focused upon below.

UTILIZATION OF ULTRASOUND

The utilization of ultrasound in ACS is multifaceted. Its usage is primarily directed at identifying the downstream effects of elevated IAP as reviewed in the pathophysiology above for diagnosis. There is also some ability to use ultrasound in therapeutic means, described below, but methods to diagnose IAH in the early stages using ultrasound are evolving. The most common methods of employing ultrasound in the diagnostic evaluation for IAH and ACS are by evaluating veins, arteries, or muscle as these are each uniquely affected by IAP.

Venous

As previously reviewed, elevations in IAP result in decreased venous return. This has been directly evaluated in studies evaluating morphology changes in the veins in elevated IAP. The inferior vena cava (IVC) size has been shown to correlate to changes in IAP. One small study of healthy human volunteers by Cavaliere and colleagues induced IAH with external compression via a pelvic stabilizer and demonstrated morphology changes in the IVC on ultrasound correlated with elevated IAP. The study also examined the right suprahepatic vein and the external iliac vein to evaluate for morphology changes in correlation with elevated IAP but did not find a correlation. The authors concluded that an IVC section area under 1 cm^2/m^2 discriminated between the presence and absence of IAH with a sensitivity of 66% and specificity of 88%.[24]

Another interesting use of ultrasound in abdominal veins in the venous excess ultrasound score (VExUS). Originally developed to evaluate for acute kidney injury after cardiac surgery, VExUS identifies venous congestion, which is also present in ACS. The score includes evaluations of the IVC, along with hepatic, portal, and renal vein Doppler patterns. Alterations in the venous flow and an IVC diameter of at least 2 cm correlate with a high risk of postcardiac surgery acute kidney injury.[25] There are multiple publications available describing the technical aspects of VExUS, and Koratala provides an excellent depiction of flow changes in response to increasing right atrial pressure (**Fig. 1**).[26] While the original development of this score was aimed at evaluating a different pathology, the concept of evaluating venous Doppler waveforms to evaluate IAH is promising. And although VExUS has not been directly studied in IAH/ACS, ultrasound evaluation of vascular congestion that occurs may have some overlap in the venous congestion findings studied in VExUS.

Arterial

The pressure from IAH is believed to impact the arteries and has been well studied in the renal arteries. Renal resistive index (RI) is defined as peak systolic velocity minus end diastolic velocity, all divided by peak systolic velocity.[27] The study referenced above by Cavaliere and colleagues also demonstrated increased RIs in the renal arteries correlated with increased IAP.[24] Another study by Chen and colleagues evaluated patients in the intensive care unit (ICU) at risk for IAH and separated them into groups (above and below 12 mm Hg) based on IAP using urinary bladder pressure. They evaluated right renal artery RI and right diaphragm thickening rate (discussed

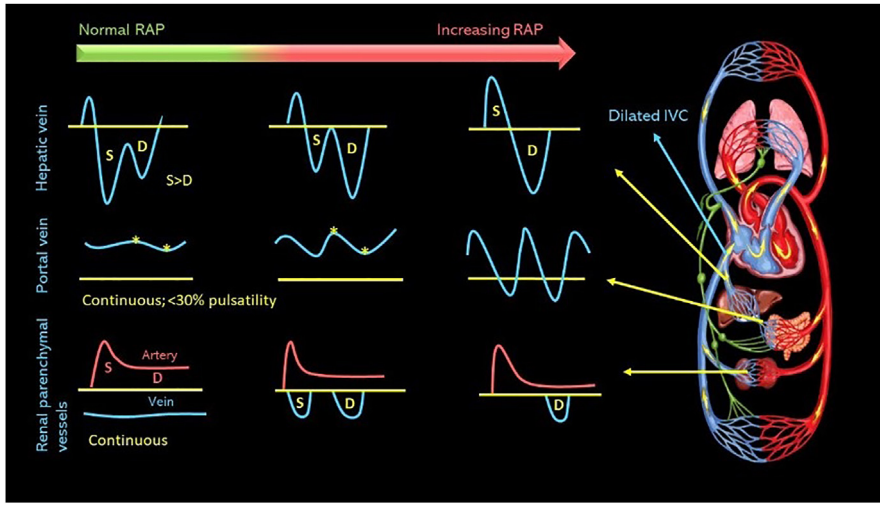

Fig. 1. Illustration showing the natural history of venous Doppler waveforms with increasing right atrial pressure. Dilatation of the IVC (typically >2 cm) usually precedes these changes. D, diastolic wave; S, systolic wave. Asterisks on the portal vein waveform indicate the highest and lowest amplitudes during a given cardiac cycle used to calculate percent pulsatility. Note that the normal portal vein waveform is above the baseline and the renal is below, which reflects the direction of blood flow toward and away from the transducer, respectively. RAP, right atrial pressure. (*Source* Koratala A. Evaluation of Venous Congestion Using Beside Ultrasonography by the Nephrology Consultant: The VExUS Nexus. POCUS J. 2022 Feb 1;7(Kidney):17–20. https://doi.org/10.24908/pocus.v7iKidney.15341. PMID: 36896109; PMCID: PMC9994295.)

below). The right renal artery RI was measured at the renal lobar artery with a convex probe. A positive correlation with IAH and increased renal artery RI was identified. This, along with the diaphragm thickening rate and lactic acid level, was used to create a predictive model for IAH. The formula is outside the scope of this article, but it is important to note the relationship of the diaphragm thickening and renal RI to IAH.[28]

Muscle

As mentioned above, the study by Chen and colleagues evaluated diaphragm muscle changes in IAH. To do this, a linear probe was used at the right intercostal space and the thickness of the diaphragm was measured at end expiration and end inspiration. The thickening rate was calculated by subtracting the end expiration thickness from the end inspiration thickness and then dividing that value by the end expiration thickness. They found a significant correlation between IAH and a decreased diaphragm thickening rate, which was used in the aforementioned model[28] The abdominal wall muscles also change in response to abdominal pressure and may be a future target for ultrasound diagnosis as well.[29]

Other Diagnostic Means

New techniques are still being evaluated for ultrasound as a diagnostic tool in IAH. For example, a small animal model was created for proof of concept to evaluate ultrasound guided tonometry in the diagnosis of IAH. IAP in increments of 5 mm Hg up to 40 mm Hg was achieved by instilling normal saline into the peritoneal cavity of swine. The authors identified decreased renal artery and portal vein flows as the

intra-abdominal pressures increased with a good correlation. Next, the authors evaluated the ultrasound guided tonometry and found that the values obtained correlated well with increasing IAP. While there are some limitations of this study, the concept holds potential for future developments with ultrasound in IAH.[30]

TREATMENT

Management

The gold standard in treatment of ACS is emergent abdominal decompression. Historically, this is achieved by opening the abdomen. However, less invasive approaches

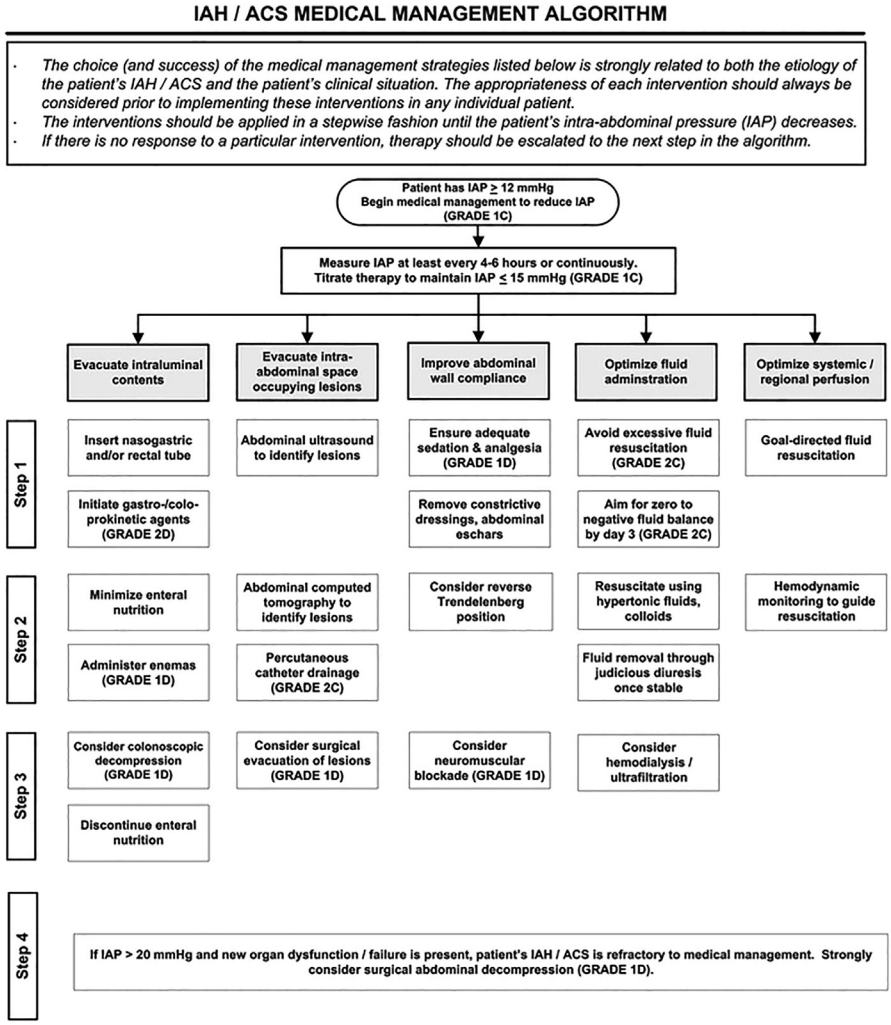
IAH / ACS MEDICAL MANAGEMENT ALGORITHM
The choice (and success) of the medical management strategies listed below is strongly related to both the etiology of the patient's IAH / ACS and the patient's clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any individual patient.
The interventions should be applied in a stepwise fashion until the patient's intra-abdominal pressure (IAP) decreases.
If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.
Patient has IAP ≥ 12 mmHg
Begin medical management to reduce IAP (GRADE 1C)
Measure IAP at least every 4-6 hours or continuously.
Titrate therapy to maintain IAP ≤ 15 mmHg (GRADE 1C)
Evacuate intraluminal contents
Evacuate intra-abdominal space occupying lesions
Improve abdominal wall compliance
Optimize fluid adminstration
Optimize systemic / regional perfusion
Step 1
Insert nasogastric and/or rectal tube
Abdominal ultrasound to identify lesions
Ensure adequate sedation & analgesia (GRADE 1D)
Avoid excessive fluid resuscitation (GRADE 2C)
Goal-directed fluid resuscitation
Initiate gastro-/colo-prokinetic agents (GRADE 2D)
Remove constrictive dressings, abdominal eschars
Aim for zero to negative fluid balance by day 3 (GRADE 2C)
Step 2
Minimize enteral nutrition
Abdominal computed tomography to identify lesions
Consider reverse Trendelenberg position
Resuscitate using hypertonic fluids, colloids
Hemodynamic monitoring to guide resuscitation
Administer enemas (GRADE 1D)
Percutaneous catheter drainage (GRADE 2C)
Fluid removal through judicious diuresis once stable
Step 3
Consider colonoscopic decompression (GRADE 1D)
Consider surgical evacuation of lesions (GRADE 1D)
Consider neuromuscular blockade (GRADE 1D)
Consider hemodialysis / ultrafiltration
Discontinue enteral nutrition
Step 4
If IAP > 20 mmHg and new organ dysfunction / failure is present, patient's IAH / ACS is refractory to medical management. Strongly consider surgical abdominal decompression (GRADE 1D).

Fig. 2. IAH/ACS medical management algorithm. World society of the abdominal compartment syndrome. (Kirkpatrick, A.W., Roberts, D.J., De Waele, J. et al. Intra-abdominal hypertension and the abdominal compartment syndrome: updated consensus definitions and clinical practice guidelines from the World Society of the Abdominal Compartment Syndrome. Intensive Care Med 39, 1190–1206 (2013). https://doi.org/10.1007/s00134-013-2906-z.)

targeting 5 categories are suggested and are outlined in a stepwise fashion by the Abdominal Compartment Society: evacuate intraluminal contents, evacuate intra-abdominal space occupying lesions, improve abdominal wall compliance, optimize fluid administration, optimize systemic/regional perfusion (Fig. 2).[5]

Point of Care Ultrasound in Treatment

Ultrasound has a role in essentially each category noted in the pathway above. It can be used to assess evacuation of intraluminal contents and evacuation of space occupying lesions. Nasogastric decompression has been confirmed and gastric contents assessed with the use of ultrasound. It has also been used to identify and evacuate space occupying lesions (ascites and intra-abdominal blood in cirrhotic patients and acute pancreatitis, respectively). When using ultrasound, a small study demonstrated the feasibility and utility of ultrasound in managing IAH.[31] Another study by Pereira and colleagues found ultrasound similarly useful in free fluid management and also in evaluating bowel contents for enema usage to lower IAP.[20]

Limitations of Ultrasound

There are many limitations in ultrasound utilization in IAH. General limitations are variability in skill and reliability between users. Using Doppler waveforms and VExUS requires a skillset that not all intensivists currently possess. Additionally, many existing studies are performed in animal models or healthy volunteers and large clinical studies are lacking. Furthermore, there are limitations in some baseline characteristics of patients. For example, obese patients often present with a baseline degree of IVC compression complicating this as a metric to assess for IAH.[32] Another example of a challenging patient population is renal arterial flow in diabetic patients. Using renal RIs can be challenging in patients with diabetes as increased RIs are common with diabetic nephropathy.[32] These are just a few limitations to highlight some considerations in employment of ultrasound in IAH.

The ability to employ ultrasound in IAH and ACS is a complex area of study that is rapidly gaining momentum with the increase in the performance of POCUS. While many of the areas are still undergoing examination, more limitations are likely to be further elucidated. However, given the invasiveness required for diagnosis and treatment of ACS, ultrasound paves a way for innovation as a quick and less invasive means.

Finally, it must be recognized that when less invasive approaches fail, the abdomen must be opened. This presents a myriad of acute and chronic challenges but may be lifesaving. Approaches to the open abdomen and its sequelae are broad and complex and are beyond the scope of this article.

SUMMARY

In summary, IAH/ACS is a complicated disease process with multiple etiologies. The ability to accurately diagnose and treat it in a timely fashion is paramount to improving outcomes. Using ultrasonography as a tool in the diagnosis and management of this disease offers a point of care technique to maximize patient care. More commonly documented techniques as described above include IVC diameter and renal artery indices; newer and less described methods include the diaphragm thickening rate and ultrasound guided tonometry. POCUS is a rapidly evolving field, and these bedside metrics can help diagnose IAH/ACS and improve patient care.

CLINICS CARE POINTS

- Abdominal compartment syndrome (ACS) is a highly morbid and even fatal process, which requires prompt recognition and intervention.
- The World Society for Abdominal Compartment Syndrome has well defined diagnostic criteria for ACS.
- Beyond prevention, there are well established published guidelines on both noninvasive and invasive management of ACS.
- Point of care ultrasound has an increasingly prominent role in both the diagnosis and management of ACS.

DISCLOSURES

The authors have no relevant disclosures that influence the content of this article.

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